functions, such as signaling and cell-to-cell interactions. Glucosylceramide synthase—encoded by the *Ugcg* gene—controls the first committed step in the major pathway of glycosphingolipid synthesis. Global disruption of the *Ugcg* gene in mice is lethal during gastrulation. The inventors have established a *Ugcg* allele flanked by loxP sites (floxed). When cre recombinase was expressed in the nervous system under control of the *nestin* promoter, the floxed gene underwent recombination, resulting in a substantial reduction of *Ugcg* expression and of glycosphingolipid ganglio-series levels. The mice deficient in *Ugcg* expression in the nervous system show a striking loss of Purkinje cells and abnormal neurologic sphingo-lipid behavior.

The Research Tools available are mice with a floxed *Ugcg* allele that can be deleted in a conditional manner. These mice carrying floxed *Ugcg* alleles will be useful for delineating the functional roles of glycosphingolipid synthesis in the nervous system and in other physiologic systems.

Applications

- Study of the functional roles of glycosphingolipid synthesis in the nervous system and other physiologic systems.
- The floxed *Ugcg* allele will facilitate analysis of the function of glycosphingolipids in development, physiology, and in diseases such as diabetes and cancer.

Development Status: Ready to Use.

Inventors: Richard L. Proia (NIDDK).

Publication: T Yamashita, ML Allende, DN Kalkofen, N Werth, K Sandhoff, RL Proia. Conditional *LoxP*-flanked glucosylceramide synthase allele controlling glycosphingolipid synthesis. Genesis 2005 Dec;43(4):175–180.

Patent Status: HHS Reference No. E–320–2007/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials license agreement.

Licensing Contact: Suryanarayana (Sury) Vepa, PhD, J.D.; 301–435–5020; *vepas@mail.nih.gov*.

Collaborative Research Opportunity: The NIDDK Genetics of Development and Disease Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the sphingolipid metabolism in physiology and disease. Please contact Dr. Proia at *proia@nih.gov* for more information.

Mutant Nuclear Orphan Receptor for Drug Metabolism Assays

Description of Technology: The constitutively active nuclear orphan receptor (CAR) activates transcription of genes encoding various drug-metabolizing enzymes, such as cytochrome P450, in response to drug exposure. While the direct activation of CAR in response to various drugs has been observed *in vivo*, CAR is always active in cell-based transfection assays, even in the absence of activating drugs. This constitutive activity of CAR makes it difficult to perform accurate *in vitro* assays to measure drug metabolism.

The NIH has obtained patent protection for modified CAR proteins that can be directly activated by drugs *in vitro*. This technology may potentially be used in the development of more efficient and cost-effective cell-based drug metabolism assays.

Applications: Development of improved *in vitro* assays to measure drug metabolism.

Inventors: Masahiko Negishi *et al.* (NIEHS).

Publications

1. T Sueyoshi, T Kawamoto, I Zelko, P Honkakoski, M Negishi. The repressed nuclear receptor CAR responds to phenobarbital in activating the human CYP2B6 gene. J Biol Chem. 1999 Mar 5;274(10):6043–6046.

2. T Kawamoto, S Kakizaki, K Yoshinari, M Negishi. Estrogen activation of the nuclear orphan receptor CAR (constitutive active receptor) in induction of the mouse Cyp2b10 gene. Mol Endocrinol. 2000 Nov;14(11):1897–1905.

Patent Status: U.S. Patent No. 7,365,160 issued 29 Apr 2008 (HHS Reference No. E–034–2002/0–US–03).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: Tara L. Kirby, PhD; 301–435–4426; *tarak@mail.nih.gov*.

Dated: January 8, 2009.

Richard U. Rodriguez,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. E9–978 Filed 1–16–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, HHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by an agency of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Use of Mono-Amine Oxidase Inhibitors To Prevent Herpes Virus Infections and Reactivation From Latency

Description of Technology: Available for licensing are methods of using Monoamine Oxidase Inhibitors (MAOIs) to prevent alpha-herpesvirus lytic infections, such as those caused by Herpes simplex virus (HSV–1 or HSV–2) and Varicella zoster virus (VZV), and to possibly prevent the periodic reactivation of these viruses from latency. MAOIs have been historically used to treat depression, hypertension, and related diseases. The invention describes how MAOIs can also inhibit LSD1, a histone/protein demethylase that is required for initiation of alpha-herpesvirus lytic infection. After an initial lytic infection, alpha-herpesviruses establish latent infections in sensory neurons and undergo periodic reactivation that results in disease ranging from mild lesions to life threatening encephalitis. Investigators have determined that MAOIs may also block the reactivation process. Due to the nature of the target LSD1 and its role in modulating chromatin modifications, these drugs could also prevent infection by or reactivation of other nuclear viruses.

Alpha-herpesviruses infections are common worldwide, with 57% to 80% of adults being seropositive for HSV. Recurrent labial herpes affects roughly one third of the U.S. population, and these patients typically experience 1 to 6 episodes per year. Genital herpes can result from infection with either HSV type and HSV–1 has become an important cause of genital herpes in

some developed countries. HSV keratitis is the most frequent cause of corneal blindness in the United States, is a leading indication for corneal transplantation, and is the most common cause of infectious blindness in the Western world.

Applications:

• Prevention and treatment of recurrent Herpes simplex virus outbreaks.

• Prevention and treatment of recurrent Varicella zoster infection.

• Treatment of HSV encephalitis.

• Treatment of Herpes keratitis.

Development Status: The investigators intend to do a series of in vivo animal studies on the efficacy of MAOIs in preventing primary infection and/or reactivation of herpes simplex virus in a mouse model system.

Inventors: Thomas M. Kristie et al. (NIAID).

Patent Status:

• U.S. Provisional Application No. 61/083,304 filed 24 Jul 2008 (HHS Reference No. E–275–2008/0–US–01).

• U.S. Provisional Application No. 61/111,019 filed 04 Nov 2008 (HHS Reference No. E–275–2008/1–US–01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Christina Thalhammer-Reyero, PhD; 301–435–4507; *thalhamc@mail.nih.gov*

Collaborative Research Opportunity: The National Institute of Allergy and Infectious Diseases' Laboratory of Viral Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of MAOIs to prevent herpes virus infections and reactivation from latency. Please contact Marguerite J. Miller at 301–435–8619 or *millermarg@niaid.nih.gov* for more information.

Method of Treating Pneumoconiosis With Oligodeoxynucleotides

Description of Technology: The inhalation of dust containing crystalline silica particles causes silicosis, an incurable lung disease that progresses even after dust exposure ceases. The World Health Organization estimates that over a million U.S. workers are exposed to silica dust annually, and that thousands worldwide die each year from silicosis. The pulmonary inflammation caused by silica inhalation is characterized by a cellular infiltrate and the accumulation of chemokines, cytokines (including TNF-alpha, IL–1, and IL–6), and Reactive Oxygen Species (ROS) in bronchoalveolar lavage (BAL) fluid.

Macrophages are the predominant immune cell type present in alveolar spaces where they play an important role in the lung pathology associated with silica inhalation. The uptake of silica particles by macrophages triggers the production of ROS (including hydrogen peroxide) via the oxidative stress pathway, which in turn contributes to pulmonary damage and macrophage death.

One potential strategy for limiting the production of proinflammatory cytokines and ROS after silica exposure involves treatment with ''suppressive'' oligonucleotides (ODN). Suppressive ODN express motifs based on the repetitive TTAGGG hexamers present at high frequency in the telomeric ends of self DNA. Previous studies showed that these motifs (released by injured host cells) block Th1 and proinflammatory cytokine production in vitro and down-modulate over-exuberant/pathologic immune responses in vivo (such as those found in septic shock and autoimmune diseases).

This application claims methods for treating, preventing or reducing the risk of developing occupational lung diseases using. Preclinical in vivo studies show that pretreatment with suppressive (but not control) ODN reduces silica-dependent pulmonary inflammation. Preclinical *in vivo* studies also showed that treatment with suppressive ODN also reduced disease severity and improved the survival of mice exposed to silica.

Application: Development of ODN-based therapeutics for the treatment of pneumoconiosis.

Development Status: ODNs have been synthesized and preclinical studies in the murine model of acute silicosis have been performed.

Inventors: Dennis M. Klinman (NCI), Takashi Sato (NCI), *et al.*

Publication: T Sato *et al.* Suppressive oligodeoxynucleotides inhibit silica-induced pulmonary inflammation. J Immunol. 2008 Jun 1;180(11):7648–7654.

Patent Status: U.S. Provisional Application No. 61/055,102 filed 21 May 2008 (HHS Reference No. E–182–2008/0–US–01)

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301–435–4646; *soukasp@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Experimental Immunology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Method of Treating Pneumoconiosis With Oligodeoxynucleotides. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Attenuated Salmonella as a Delivery System for siRNA-Based Tumor Therapy

Description of Technology: The discovery that genes vectored by bacteria can be functionally transferred to mammalian cells has suggested the possible use of bacterial vectors as vehicles for gene therapy. Genetically modified, nonpathogenic bacteria have been used as potential antitumor agents, either to elicit direct tumoricidal effects or to deliver tumoricidal molecules. Bioengineered attenuated strains of *Salmonella enterica* serovar *typhimurium* (*S. typhimurium*) have been shown to accumulate preferentially greater than one-thousand fold in tumors than in normal tissues and to disperse homogeneously in tumor tissues. Preferential replication allows the bacteria to produce and deliver a variety of anticancer therapeutic agents at high concentrations directly within the tumor, while minimizing toxicity to normal tissues. These attenuated bacteria have been found to be safe in mice, pigs, and monkeys when administered intravenously, and certain live attenuated *Salmonella* strains have been shown to be well tolerated after oral administration in human clinical trials. The *S. typhimurium* phoP/phoQ operon is a typical bacterial two-component regulatory system composed of a membrane-associated sensor kinase (PhoQ) and a cytoplasmic transcriptional regulator. phoP/phoQ is required for virulence, and its deletion results in poor survival of this bacterium in macrophages and a marked attenuation in mice and humans. phoP/phoQ deletion strains have been employed as effective vaccine delivery vehicles. More recently, attenuated salmonellae have been used for targeted delivery of tumoricidal proteins.

This technology comprises live, attenuated *Salmonella* strains as a delivery system for small interfering double-stranded RNA (siRNA)-based tumor therapy. The inventors' data provide the first convincing evidence that *Salmonella* can be used for delivering plasmid-based siRNAs into tumors growing in vivo. Claimed in the related patent application are methods of inhibiting the growth or reducing the volume of solid cancer tumors using the si-RNA constructs directed against genes that promote tumor survival and cancer cell growth. The Stat3-siRNAs carried by an attenuated *S. typhimurium*

described in the application exhibit tumor suppressive effects not only on the growth of the primary tumor but also on the development of metastases, suggesting that an appropriate attenuated *S. typhimurium* combined with the RNA interference (RNAi) approach may offer a clinically feasible method for cancer therapy.

Application: Development of live attenuated bacterial cancer vaccines, cancer therapeutics and diagnostics.

Development Status: Vaccines have been prepared and preclinical studies have been performed.

Inventors: Dennis Kopecko (FDA/CBER), DeQi Xu (FDA/CBER), *et al.*

Related Publications:

1. L Zhang *et al.* Intratumoral delivery and suppression of prostate tumor growth by attenuated Salmonella enterica serovar typhimurium carrying plasmid-based small interfering RNAs. Cancer Res. 2007 Jun 15;67(12):5859–5864.

2. L Zhang *et al.* Effects of plasmid-based Stat3-specific short hairpin RNA and GRIM–19 on PC–3M tumor cell growth. Clin Cancer Res. 2008 Jan 15;14(2):559–568.

Patent Status:

• Chinese Patent Application No. 200610017045.5 filed 26 Jul 2006 (HHS Reference No. E–278–2007/0–CN–01).

• PCT Patent Application No. PCT/US2007/074272 filed 24 Jul 2007, which published as WO 2008/091375 on 31 Jul 2008 (HHS Reference No. E–278–2007/0–PCT–02).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301–435–4646; *soukasp@mail.nih.gov*.

Collaborative Research Opportunity: FDA–CBER Division of Bacterial, Parasitic, and Allergenic Products is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize *Salmonella*-delivered anti-tumor therapies or *Salmonella*-vectored vaccines. Please contact Alice Welch at *Alice.Welch@fda.hhs.gov* for more information.

Dated: January 8, 2009.

Richard U. Rodriguez,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. E9–979 Filed 1–16–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the meeting of the National Cancer Advisory Board.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

A portion of the meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4), and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Cancer Advisory Board; Ad Hoc Subcommittee on Communications.

Open: February 2, 2009, 6:30 p.m. to 8 p.m.

Agenda: Discussion on cancer communications.

Place: Bethesda Marriott Suites, 6711 Democracy Boulevard, Bethesda, Maryland 20817.

Contact Person: Dr. Paulette S. Gray, Executive Secretary, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Room 8001, Bethesda, MD 20892–8327, (301) 496–5147.

Name of Committee: National Cancer Advisory Board.

Open: February 3, 2009, 8 a.m. to 4 p.m.

Agenda: Program reports and presentations; business of the Board.

Place: National Institutes of Health, 9000 Rockville Pike, Building 31, C Wing, 6th Floor, Conference Room 6, Bethesda, MD 20892.

Contact Person: Dr. Paulette S. Gray, Executive Secretary, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Room 8001, Bethesda, MD 20892–8327, (301) 496–5147.

Name of Committee: National Cancer Advisory Board.

Closed: February 3, 2009, 4 p.m. to 5 p.m.

Agenda: Review of grant applications.

Place: National Institutes of Health, 9000 Rockville Pike, Building 31, C Wing, 6th Floor, Conference Room 6, Bethesda, MD 20892.

Contact Person: Dr. Paulette S. Gray, Executive Secretary, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Room 8001, Bethesda, MD 20892–8327, (301) 496–5147.

Name of Committee: National Cancer Advisory Board.

Open: February 4, 2009, 8 a.m.to 12 p.m.

Agenda: Program reports and presentations; business of the Board.

Place: National Institutes of Health, 9000 Rockville Pike, Building 31, C Wing, 6th Floor, Conference Room 6, Bethesda, MD 20892.

Contact Person: Dr. Paulette S. Gray, Executive Secretary, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Room 8001, Bethesda, MD 20892–8327, (301) 496–5147.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

In the interest of security, NIH has instituted stringent procedures for entrance onto the NIH campus. All visitor vehicles, including taxicabs, hotel, and airport shuttles will be inspected before being allowed on campus. Visitors will be asked to show one form of identification (for example, a government-issued photo ID, driver's license, or passport) and to state the purpose of their visit.

Information is also available on the Institute's/Center's home page: *deainfo.nci.nih.gov/advisory/ncab.htm*, where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: January 9, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–996 Filed 1–16–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Research Resources; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meetings.